FILE NAME: Saranac 7th Symposium (SSY)

DATE: 1952

DOC#: SSY015

DOCUMENT DESCRIPTION: Transcript of Presentation by Dr. Friedman - Pneumoconiosis in Coal Miners in Alabama

TAKEN FROM PAGE 257.

PNEUMOCONIOSIS IN COAL MINERS (Continued) 2:30 to 5:30 P.M. Sept. 23, 1952.

Chairman: O. A. Sander, M. D.

Pneumoconiosis in Coal Miners in Alabama

Louis Friedman, M. D.

Discussion

Pulmonary Function Studies of Coal Miners in Pennsylvania and West Virginia

Burley L. Motley, M. D.

Pulmonary Function Studies of Coal Miners in Wales

Philip Hugh-Jones, M. D.

Discussion

BY DOCTOR SANDER:

Let's have the meeting come to order. We'll carry

on with Black Tuesday, the Pneumoconiosis of Coal Miners day, and see if we can confuse and confound you a little more this afternoon. First of all, doctor Vorwald has an announcement.

BY DOCTOR VORWALD:

It's a very simple one, and that is we are requested again to remind you, those of you who wish to attend the banquet, to please register before tomorrow noon for the simple reason that the hotel manager must have the number of people who are coming so that he can prepare the adequate amount of food for the group and set up the number of tables that might be requested, so please register for the banquet and know also that the ladies accompanying you are invited to the banquet.

BY DOCTOR SANDER:

The first speaker this afternoon has had a large clinical experience with a group of bituminous miners in Alabama. He will tell you about it, his clinical experience with this group. Doctor Louis Friedman of Birmingham.

BY DOCTOR FRIEDMAN:

My remarks this afternoon will be based upon our experiences in the study of about three thousand soft coal miners in Alabama. When I was much younger and before I had the advice and counsel of people like Doctors Sander, Vorwald and Doctor Lanza and Doctor Hussey, I accidentally

TAKEN FROM PAGE 258.

By DOCTOR FRIEDMAN:

My remarks this afternoon will be based upon our experiences in the study of about three thousand soft coal miners in Alabama. When I was much younger and before I had the advice and counsel of people like Doctors Sander, Vorwald and Doctor Lanza and Doctor Hussey, I accidentally

called my disease 'anthraco-silicosis', with some justification, I thought. They have subsequently persuaded me, energetically and in a very friendly manner, to convert the title of my paper to 'Pneumoconiosis in Soft Coal Miners' and then when I mentioned that to Doctor Lanza, he added 'In Alabama'. I had nothing to do with the title.

The pulmonary dust disease, as we see it in soft coal miners, is a disease which produces disability, both on a structural and functional basis. My remarks to you this afternoon will be from the point of view of a clinician as Doctor Sanders indicated, and our great concern is the correlation of X-ray findings, clinical history and functional studies with the total picture presented to us by the patient.

Unfortunately, it is not always possible to describe disability on the usual standards in the case of coal workers' pneumoconiosis. Only so frequently do we find a lack of Roentgenographic findings and nevertheless, we are confinced that the man's occupation had something to do with his present disability.

I would like to show you -- slide, please -- in the first slide, I would like to show you some of the terminology that we use in Alabama, at least we use in our clinic, to simplify the problem. Now, in presenting this classification of clinical disability and Roentgenographic

classification of the disease, I do not mean to take issue with Doctor Fletcher in the wonderful work that he has done in breaking this Roenggenographic picture down to its fine elements. To me, this is the clinician's idea of the classification in which you ascribe very little, in which you do not rely upon your X-ray for further than identification, and in some instances only do you use it on the basis of very pronounced structural changes, to determine the fact that this man must obviously be disabled. We merely use the Roentgenogram to classify the particular disease and call it pneumoconiosis of soft coal workers, and then we use our clinical studies and laboratory studies to determine whether there is any disability.

We consider, in sensing a man's disability, that a man is either disabled or not disabled as a result of his occupational disease, and on that basis, you can have a disease which is disabling, temporarily disabling, as I will show you later in the slides, or you can have a disease which is non-disabling, and for those of us who so frequently find it necessary to sit on a fence, we have the fourth classification of possibly disabling disease, until we can make up our mind three months later when you come back for another X-ray and examination.

From the point of view of assessing disability, I would like to take this moment to say it is very easy for

a doctor to rely upon his function studies after they're done in the proper fashion and say 'This man has forty percent disability'. Well, that is from a medical point of view, but you take a coal miner who has no education and who has worked in a coal mine all of his life and at the age of forty-five, fifty or fifty-five, suddenly finds himself with a disease that will progress is he is exposed to his present occupation for any further time, to my way of thinking, and I think that is shared by some of the members here today, that man is actually a hundred percent disabled, because he can no longer find his economic place in society to gain a livelihood and take care of himself and his family.

Now, as regards the Roentgenographic classification, you will notice that we have Type 1, which is a linear variety, a linear change in the X-ray which probably corresponds to Doctor Fletcher's simple pneumoconiosis, and then the Type 2, the nodular variety which we do not see very frequently in coal miners, and Type 3, the mixed, one in which you see a picture of linear changes and nodular changes in the Roentgenogram, Type 4, the conglomerate coalescent variety, maybe a progressive change from Type 1, 2 or 3, so you may have conglomerate coalescent nodular pneumoconiosis of soft coal workers, and finally in Type 5, the Roentgenogram itself, you can see that this is a

complicated case, pneumonia, bronchiectasis, spontaneous pneumothorax or some other complication which we are able to discern from the Roentgenogram and, therefore, in sending a communication to another doctor, another clinician, who doesn't get too deeply into the breakdown of Roentgenographic types of disease, we could say that this man has non-disabling linear pneumoconiosis of soft coal workers and immediately he knows what we're talking about.

Next slide -- No, on this first slide, you'll see -- I wonder if we put the lights off whether we couldn't have a better view of the screen. Now, on this first slide you see the X-ray of a twenty-seven year old boy who died of accute monocytic leukemia, after working only three and a half years in a soft coal mine, and if you notice his Roentgenogram, there is nothing particularly revealing about it and it could be classified as increased bronchiovascular markings.

However, the next slide -- you skipped -- Now, this slide shows a cross-section of his lung prepared by Doctor Gough in Wales, and you will see that he already has the deposition of coal nodules throughout his entire lung. Now, I use the term coal macules to correspond with Doctor Hebbleston's description of the disease pathologically. Now, if you will notice, although he has these

marked macules throughout his entire lung field, there is no evidence of nodulation in the X-ray and we have no evidence from the X-ray that this man even had any changes compatible with an early type of pneumoconiosis. However, those of us who work with the disease and know that the changes, the early changes or the nature that we saw of the X-ray of the patient, would suspect that something was present in this man's pulmonary tissue that wasn't present in a normal individual's chest.

Now, this is an example of another linear variety of pulmonary dust disease in soft coal workers and, although this patient doesn't look particularly disabled from Roentgenogram, he is unable to participate in any gainful occupation after having worked thirty-seven years as a coal worker in Alabama.

Next slide. Now, this is a slide also showing only increased bronchovesicular markings and nothing particularly significant. This man died of accute lymphatic leukemia after having worked thirty-five yearsin a soft coal mine. I would like for you to look at the apices of this lung very carefully, because the whole lung section in this case will not correspond at all with what you have seen from this man's X-ray.

Here you see another whole lung section prepared by Doctor Goffe, and you see the focal emphysema, the coal

deposits and some evidence of silica spread throughout this man's lung field. Again, pointing out to us very clearly that you can not rely upon the Roentgenogram to determine either function or the degree of pathology which you will find when you examine the tissue at necropsy.

Now, this is an example of nodular type of pneumoconiosis in the soft coal worker. That's what you think and that's what I thought, but it isn't so. This man worked in the coal mine, but he actually wasn't a coal miner. He worked on a motor up a haulage way and for thirty-three years, he sanded a track in front of him and he was inhaling pure silica during that entire period of time and it's only in those instances in Alabama that we are able to find both a motorman and people who do a lot of drilling in hard rock, evidences of the nodular variety of pneumoconiosis in soft coal workers, so actually this man, although he worked in a coal mine really wasn't a coal miner and he actually has a true case of silicosis.

Now, this is an example of a seventy year old man and, as you see, he has both nodules and linear changes in both lung fields symmetrical, and you would think from this X-ray, that the man should have a great deal of disability. On the other hand, he never complained of any signs and symptoms referable to his - to his lungs, no dypsnea, no cough, and he died of a carcinoma of the stomach.

I want to apologize for the fact that this was not prepared in color, but as you see in this whole lung section interpreted by Doctor Goffe, indicates that this man not only had coal but a great deal of silica in his lung tissue. At this point, I would like to say that we have two groups of patients in our clinic. We have coal workers and ore miners and, in our experience, it appeared that the coal miners suffer greater disability from their exposure to coal dust than do people who work in ore mines.

In this particular case, you can see the extensive changes in this man's lung field without any evidence of clinical disability for a man of his age compared to another man who had to work in a coal mine of the same age. The next line is the microscopic section and you see here, you see the silica nodules, you see some of the silica deposited in whirls and in the middle of the nodule, you see the coal dust.

The next slide. Now, this is an example of a Negro who had worked twentym some years in a coal mine who came in complaining because of pain in his chest. We examined him very carefully and even though we maintain a high index of suspicious - of suspicion that this man may be suffering from pneumoconiosis of soft coal workers in Alabama, we found no evidence to substantiate our clinical impression that he may have the disease. Therefore, we did

some additional studies and in the esophageal examination with berium, revealed a little nick in his esophagus and because he had a widened mediastinum, we decided to explore this man and see what his trouble was.

Well, that surgery, Doctor Charles Donald, a thoracic surgeon, performed this operation, found that both of his lung fields were full of nodular masses which we were unable to see in a Roentgenogram, and this microscopic section was taken from the mediastinal lymph node which was the size of a tennis ball, and as you see in this mediastinal node, evidences of silica in whirls and coal deposits, so this was another example of more pathology being present in the pulmonary tissue than we were able to discern in the Roentgenogram.

Now, this is an example of a man who died in his seventies, and of a cerebrovascular accident. He suffered from hypertensive arterial cardiovascular disease, and if you notice his Roentgenogram, examine it carefully, you don't see anything too revealing. You see the evidence of increased markings and for most of us who are really interested in disease we could possibly read into this focal emphysema.

Now, notice the left lung and notice the right lung and notice the next slide that appears. Focal emphysema, evidence of the coal macule degenerating and causing

the focal emphysema and similarly, in the left lung over there, so you see again the evidence of the disease pathologically is much more extensive than you can discern in the Roentgenogram.

Now, this is only a clinical example of what we term conglomerate coalescent pneumoconiosis in soft coal workers. Here you see a solitary nodule in the right apex. Now, I would like to say this, in the ensuing slides, you will see more examples of conglomeration and coalescence and we experience a great deal of difficulty in attempting to prove the diagnosis of tuberculosis in these patients.

By very exhaustive laboratory studies which include smears, concentrates, cultures, guinea pigs, and all other varieties of tests which may ascertain the presence of tuberculosis - of tubercle bacillus, we only too frequently find that we are unable to demonstrate it. Perhaps Doctor Vorwald's contribution last year or the year before on the B. C. G. vasicine in silicotic animals could possibly explain the fact that we have evidences of infection or at least evidences of conglomeration, and coalescence in those places in the lung where you ordinarily find tuberculosis, and still we are unable to find a tubercle bacillus. Perhaps it doesn't take too many tubercle bacillae to set the process in motion here and later on, we just can't find them either in life or in death. As a matter of fact, during

life, we only find tuberculosis in our patients in about ten or fifteen percent of the cases, whereas we're able to double that at the necropsy table.

This is another example of conglomeration and coalescence in a soft coal miner, and again we were unable to prove the existence of tuberculosis, and incidentally, in this particular case, the man's tubercular test was negative. Recently, I have come to the conclusion, whether it's right or whether it's wrong, that whenever you have conglomeration and coalescence in assymmetry in the lung fields of people who serve - who suffer from pneumoconiosis, I believe that we are justified in ascribing the assymetry of the conglomerate and coalescent lesion to a superimposed infection, more than likely tuberculosis.

Next slide. This, now, is an example of a fifty-five year old coal miner who has bi-lateral conglomeration and coalescence which probably fits the terminology of angel-wing distribution of the disease. This man had open hemoptysis - the man had hemoptysis on frequent occasions and we examined his sputum on the time that he had hemoptysis and the times he didn't have hemoptysis and we have still been unable to prove a tuberculous infection in this particular case. Of course, he's terribly disabled, not only structurally from what we see in the Roentgenogram, and what we know will happen to the man, but also on the

basis of functional studies.

In the next slide you will see what I presume to be an ensuing stage of this man's disease. Here you see cavitation in the right upper lobe and a similar type of lesion in the left lung as we demonstrated in the previous slide. Of course, we had no difficulty in this particular case in proving the existence of tuberculosis, tuberculosis in this patient.

Next slide. I believe that the slide before the one I just showed can go to either the tubercular stage that I just showed you or else it can develop the conglomerate coalescent features of the X-ray on the right or it may go on to develop the changes noticed here on the left, in the left slide, the left X-ray.

It may be of interest -- one moment, please -- it may be of interest to you to know that both of these patients at autopsy had tuberculosis and exhaustive studies in both instances over a period of several years, failed to reveal the presence of tubercle bacillae, and at the autopsy table, the tissue obtained was examined by Doctor Casey, the pathologist in our community. He had a great deal of diffuculty in finally obtaining sufficient evidence to call this tuberculosis, but he did find the organism.

Next slide. Now, this is an example of another coal miner who worked twenty-nine years in a coal mine --

excuse me -- twenty-six years in a coal mine and three years in an ore mine. Notice the conglomerate and coalescent lesions in his apices. This is not an unusual picture in Alabama, where our coal miners have to do a lot of drilling, so we have a sufficient admixture of coal dust and silica to present this type of picture. I want you to notice the lower parts of both of these lung fields and the upper parts and then in the next slide, you can see the conglomerate coalescent lesion in the pathology and in the lower half of the lung field, you see nodules or macules of coal which were not discerned in the Roentgenogram, indicating again that the coal macule itself is not the radiopaque - has not the radiopaque character which we ascribe to silica or fibrous tissue, and if you were to rely completely on the Roentgenogram, you would miss so many cases of pneumoconiosis in soft coal workers.

Additionally, I want you to notice the focal emphysema in the lower half of this man's lung fields.

Now, this is an example of pneumoconiosis in a soft coal worker with cavitation in the right upper lobe. I was convinced, as well as everyone else, that saw this case, that this patient had tuberculous infection superimposed, but instead he turned out to have -- next slide -- what Doctor Bolliter has desdribed as a sceptic necrosis. We were able to find the cavity, but we were never able to

find any tubercle bacillae. Again I want you to notice the extent of symple pneumoconiosis in the right lobe and again in this lung over here, notice the large amount of coal deposits and the cavity without any evidence of tubercle bacillae, both in life and in death at the necropsy table, and very careful studies.

Next slide. Now, the problem frequently comes up, and the question is often asked, is pneumoconiosis a progressive disease? I think that in this slide and in ensuing Roentgenograms, I can say and show you that it is. If you notice, this first slide, incidentally, all of the whole lung sections in pathology which I am showing you today have been prepared by Doctor Goffe in Cardiff, Wales and he lent me these tissues so I could demonstrate them to you.

If you notice here, in this first whole lung section, there are coal macules and in this lung we find no evidence of focal emphysema. However, in another case which had been a coal miner for a longer period of time, here you see the generating coal macules and focal emphysema and some coal macules maintain themselves without degeneration.

Now, what the contracture of the coal macule is due to is only a matter of conjecture. It may be that the impure fractions in coal dust besides carbon, have a fibergenic tendency and cause this contracture over a long period

or else it may be due to the mechanical effect of the macule and the effect on the macule of the surrounding tissue. Perhaps this man had some silica in his coal macule, just enough to cause the fibrous contracture over a long period of time.

And now you see, in this final section here, all of the coal macules have disappeared and the entire lung field represents a Swiss cheese affair of focal emphysema. This, I believe indicates pathologically at least, that the disease is progressive, and you can see the progression in one slide, that the middle lung section - within that one section, you can see the focal emphysema and the macule before it has degenerated. While we're looking at these sections, we may see the reason why so many of the Roentgenograms appear to have only increased markings.

Of course, most of this, most of the macules and the tissue, as we see it, is not radiopaque and, therefore, in a large - in an X-ray of the entire lung, you have what may be called a little out effect, increased areas of translucency and increased areas of opacity, but they neutralize themselves when they finally come to the X-ray screen and that's why we see only evidences of linear markings, and of course, some of these linear markings may be ascribed to what Doctor Vorwald pointed out this morning in his microscopic sections.

Next slide. Now, this is a man who left the - left his coal mining occupation in 1948. If you notice the first X-ray, this was taken in 1949. Notice the type of X-ray he has. At this time, the man had already left his occupation in the coal mine and had no longer been exposed to any coal dust.

However, in the second X-ray taken one year later, you see that he has developed conglomeration and coalescence in areas where previously it was only suggested, that it had just started and then finally, in this last X-ray, another year later, you see that he has had additional progression of his disease.

Now, whether the progression of the disease in this case is due to the effect of the coal dust upon the tissue, or whether it is due to the fact that he had a silica admixture in the dust that he breathed, or whether it is due to superimposed infection, is not of material concern. The fact of the matter remains that this man removed from his occupation, showed a progression of disease in his lung fields which may be ascribed, directly or indirectly, to the basic pneumoconiosis.

Next slide. Now, in dealing with these patients, we find certain complications which are more frequent tahn others and if you notice the first complication listed is emphysema, the second one, bronchitis and bronchiolitis,

and then below I explain those two.

Emphysema and bronchitis are so constantly associated with clinically significant disease that they should be probably considered part of the clinical entity rather than complications. Now, we also have recurrent pneumonia in these patients, cor pulmonale is very prevalent with associated cardiac decompensation. As a matter of fact, if the cardiologist would examine people who suffer from pneumoconiosis, they would not regard cor pulmonale as an unusual entity, butwould find it not only has accute manifestations, but we who are interested in dust disease of the lung find it is quite prevalent as a chronic disease just like hypertension.

Bronchiectasis, we have found in a substantial number of our patients; tuberculosis is, of course, one of the complications. Spontaneous pneumothorax, we have observed about twenty to twenty-four patients; I don't know the exact number, in our series, and I think that's a higher incidence than you should find in the control population group.

And, finally, pulmonary osteoarthropathy, we do find and I put that down as a complication because of the manner in which you can see dramatic results, when you have this hypertrophic pulmonary osteoarthropathy, if you treat the man's lungs, clear up the bronchiectasis, the bronchial

edema and infection, and don't give him any aspirin for - or any specific measures for his arthritis, so frequently is there a manifestation of improvement in his pulmonary function, general pulmonary function, the effects of the pulmonary osteoarthropathy is gone, with few exceptions for inclement weather and so on, as long as you maintain a rather satisfactory situation in the man's lungs.

Next slide. Now, this is an example of a patient who had pneumonia and then - now, of course, this man expectorates at a rate of sputum daily that I thought he had bronchiectasis, but you can see from the bronchogram, it is within normal limits and in the right lower lobe where it doesn't fill, probably the result of accumulated secretions, indicating that these people do have an entity which may be confused with bronchiectasis and has to be differentiated from the bronchiolitis which is associated, probably responsible for the bronchospasm and the bronchial edema, and a lot of these - a lot of disability, which one sees in these cases.

When you clear up the bronchiolitis, these patients have experienced a great deal of relief, and are able to do many things that they haven't been able to do previously.

Next slide. Now, this is an example of what we call temporarily disabling pneumoconiosis in soft coal

workers. In his lower left lung you'll see he has pneumonia and then there you see about a month later or so, that the area of pneumonia has cleared. Now, this patient, when we first saw him was critically ill, and such is the case with all of our patients who develop secondary complications. They live in such a delicate cardio-respiratory balance that a supervening, intervening infection, pneumonia or systemic infection throws the weight of balance against them, and these people become critically ill and disabled.

However, when they're treated and they get over their illness, they return to their previous status, perhaps with some additional damage to their pulmonary tissue which we can't measure and want to resume their work.

Incidentally, this patient is back at work in a coal mine and does not want anybody to know about the fact that he has pneumoconiosis because if they find out, they will take him off his job, which should be done. He has been advised to leave his occupation, but we as doctors, have no right to say to a man, 'You get out'. It's up to him to make the final decision, because he's the one who has to survive.

Incidentally, I only want to mention one thing about the treatment of these patients. You can not rely upon the parental administration of antibiotics and other agents. These people, to obtain the best results

therapeutically, I don't care what you use as long as you use something that's indicated and that has been successful in your hands. Parental therapy with penicillin, for example, in a case like this, would prolong the patient's convalenscence maybe two or three months before you find that he is able to go to work, whereas with aerosol therapy, you attack the disease directly either by hand or with intermittent breathing pressure unit designed by Doctor Motley. It doesn't make any difference, but remember, the aerosol route, the direct approach to the disease.

Next slide. Now, this is an example of an individual with cor pulmonale. Notice the lacy character of the changes in his lung fields. He worked in a coal mine for thirty-five years. He is seriously disabled and he is unable to take a bath.

Incidentally, I'd like to point out that in our clinical experience, we find two very important things to ask patients, because sometimes a patient comes to you because he knows that the disease is compensable and he comes with the idea that you're going to establish the diagnosis and he comes in complaining of shortness of breath, cough, can't work, can't sleep, can't do nothing, is mad at his wife, nervous, can't do anything, but there are a few things you can find out.

For one thing, these people can't take a bath in

hot water. If they take a lot of water, and if they really have pulmonary disability on the basis of their pulmonary disfunction, they become more short of breath and have to be helped from the tub. That's due to peripheral vasal dilitation and increased oxygen demand.

Secondly, these people are always more short of breath in the morning than they are at any other time of the day except when they're working, so after they've had a good night's rest, when they wake up in the morning, if you ask them how do you feel, if they're really suffering from a dyspnea due to pulmonary disease, they'll tell you 'Doctor, I didn't sleep well and when I got up this morning I didn't feel well; it took me about an hour to stir around and start feeling better'. That's a good differential diagnostic point which we should keep in mind and it would also help us to differentiate between cardiac disease - dyspnea and pulmonary dyspnea.

Next slide. This is an example of cardiac failure in a patient with pneumoconiosis and before you - and before you label this patient as having pneumoconiosis, you should correct his heart failure first and then re-examine his roentgenogram to see if he still has evidences of pneumoconiosis. Here you can see the enlarged cardiac shadow on the left with increased markings of nodularity, and then here on the right you find that the cardiac shadow is

much smaller but he still has the changes in both lung fields compatible with pneumoconiosis.

In the management of these patients, it is important to remember that you do not treat these patients with cardiac failure the way you do someone who has cardiac failure on the basis of rheumatic heart disease or arterial sclerotic disease. In these people, it is important to correct the deranged pulmonary function and make some attempt to correct first before you digitalize him, otherwise you are apt to create a load on the right side of the heart and precipitate a fatal accident.

Next slide. I don't know how this got here, but it is. Would you mind taking this out and leave it for last. This is a joker. Now, this is an example of a patient with pneumoconiosis, heart failure and bronchiectasis, all of those things in one patient. Notice the dilated bronchi in the lower lobes and notice the cardiac failure in the middle picture. This patient is still living. He lives in an oxygen tent, has been living in an oxygen tent for over two and a half years.

Next slide. Now, this is an example of chiefly linear increased markings with some evidence of nodulation and a spontaneous pneumothorax on the left, on the left-hand side. Now, when I say a man has linear pulmonary dust disease, or when I say a man has nodular pulmonary dust disease

or mixed, I meanthat it's predominantly linear, that it's predominantly nodular and when it isn't predominantly one or the other, I call it mixed, because as a clinician, we have to have a simple method of approach to this problem that you can present to other doctors in the community, to doctorw who work in coal mining camps, so that they can work with you and understand what you're trying to accomplish, and when you get too scientific with them, when you get into the category of too much research, with a little doctor who works in the country, you lose all of his cooperation, so we have to give them something that we can see and that's simple.

Next slide. This man lived a little over three years, a little over three years in an oxygen tent and then died looking like a skeleton and if you see, in this lung field, he has evidence not only of silicosis, but also over here you see evidence of anthraco -- excuse me - of pulmonary dust disease of coal miners, with focal emphysema and macule, -- habit will always come out.

Now, next slide. Now, here you see the X-rays of two individuals who have about the same extent of pulmonary change in the Roentgenogram. Now, the sixty-four dollar question, how can an X-ray man tell me which one is and which one is not disabled? But just to speed things,up, I'll tell you that the man on the left is disabled and the man on the

right is not. However, I want to tell you that subsequently the man on the right has developed disability, but at the time of this examination, he was not disabled.

Next slide. Now, this is an example of extensive changes in the pulmonary field, in a patient forty-five years old, twenty-nine years a coal miner, no disability. Nevertheless, look at the extensive changes in his lung field, by comparison with some of the other Roentgenograms which you have seen both in this lecture and in the other lectures which preceded mine.

Next slide. Here is an example of a seventy-year old man with an elevated - with an elevated leaf of the diaphragm on this side, resulting from pleurisy, pneumonia, several years previous to this Roentgenogram. He is seventy years old; he works; he worked in a coal mine at the time this Roentgenogram was taken and he had absolutely no disability that we could find, either - either in the clinic, on clinical basis or in a laboratory.

Next slide. Now, this is an example of tissue reaction. These two boys worked alongside of each other for fourteen years. This man is thirty-six years old, I think, and this one is thirty-four years old. Notice the difference in the way these tissues have reacted. Here you have a nodular variety of change in this man's lung field and in the one on the right, has chiefly a linear change in his lung

field, indicating in this case that two people of different stock, working in the same occupation, reacted differently.

Next slide. Now, here is an example of two boys who worked alongside of each other, I think, for thirty some years. You notice the similarity of their Roentgenographic changes.

Next slide. Here are two other brothers, both with negative tubercular tests, both with negative tuberculin test, and we can't prove tuberculosis. They worked nineteen years together and they've worked about ten years apart and you can't, well, you know, you have to say they look alike anyway, and it's just remarkable to see the similarity of the changes in these two X-rays even though they worked ten years at another occupation.

Next slide. Now, these two, these two brothers are the first of four brothers. Now, notice how similar these two X-rays appear. These boys worked together forty-one years. Notice the similarity and the change in these Roentgenograms, but they're brothers, and now their other brothers worked in another part of the same coal mine for thirty-one years together. Notice the man on the right with the conglomeration and coalescence and the one who has only worked about fifteen years already beginning to manifest the changes of his brother alongside of whom he worked.

Next slide. You got the last one - you got the

one you put up the first time - I asked you to put it last? And now, just to confuse you, as well as I'm confused, this is two brothers. These two brothers worked alongside of each other -- excuse me, not alongside of each other, they worked for thirty-seven years on opposite sides of a railroad car. They did loading and the reason they worked on opposite sides of the railroad car were because they loved each other and they wanted to be sure in case a rock would start falling on the brother, they would see it and they'd run over and save the brother.

Look at these Roentgenograms. See how different they are. Worked on opposite sides of a coal car, so therefore, even though Doctor Lanza is much older than I am, I'm going to improve on his terminology. I'm going to say from now on when you talk about pneumoconiosis, you have to talk about pneumoconiosis in soft coal workers in Alabama in Jefferson County in Mine Number 9, Shaft Number 8 on the right-hand side of the railroad track, and that's about what it comes down to.

Now, in conclusion - in conclusion, I think I have about two minutes left, is that right, Doctor Sander?

BY DOCTOR SANDER:

One.

BY DOCTOR FRIEDMAN:

That always happens when you deal with Yankees.

In conclusion, I'd like to remark as follows: That there is a disease in coal workers, that it's due to their occupation, that the disease, once it manifests itself clinically, is progressive, that different people may react differently to the same exposure of dust over the same period of time. As a rule, I have sixteen pairs of brothers - that's why I say as a rule - as a rule, brothers will react similarly, but you can't depend upon it, because I just showed you.

(Applause).

BY DOCTOR SANDER:

Well, as you can see, Doctor Friedman has had a considerable clinical experience with this coal miners disease. I have asked Doctor Fletcher to open the discussion of Doctor Friedman's remarks, and then I would - I'm going to ask for questions from the floor after Doctor Fletcher has finished his discussion, and I hope you'll have a good many questions to shoot this way.

BY DOCTOR FLETCHER:

Doctor Sander has said that I could make one preliminary remark on my paper this morning, which I neglected to say this morning. I suggested that if we could abolish tuberculosis in mining areas we could abolish the worst effects of disease. That might be taken to mean that pneumoconiosis is a form of tuberculosis. I just want to say that any such interpretation of my remarks is the exact opposite

of what I mean. The expression of this disease is by dust suppression. It is an occupational disease; it is not tuberculosis, but if the utmost human endeavor fails to reduce dust levels, that in which no radiological activity provails, then the lessening of tuberculosis on this disease, might be lessened by strict tuberculosis control in this area. I am glad I have an opportunity to remove any misapprehension in regard to my remarks.

In regard to Doctor Friedman's excellent paper, I really want to sympathize with him from the bottom of my heart, because I started studying this disease as a clinician and it wasn't until I had been at it as a clinician for sometime that I managed to get my epidemiological colleagues to show me something about the disease.

As a clinician one is grossly limited. He tells us that he sees spontaneous pneumothorax as a common complication of this disease. That means to say that patients who have spontaneous pneumothorax with this disease come to his clinic. It doesn't tell us anything about the frequency of that complication in the community of men with pneumoconiosis, and we clinicians are at a gross disadvantage in studying occupational disease in that we can not resist analyzing our figures and thinking that they may mean something. It's a temptation that I have had to strenuously resist myself, under the most tremendous barrage of criticism

from Doctor Cochran, my epidemiological colleague, who thinks of clinicians with - as one of the lesser breed of rodents.

So I just want to sympathize with Doctor Friedman and congratulate him that he has derived so many interesting conclusions to the severity of this disease. I would just like to quarrel with him about this temporarily disabling pneumoconiosis. He says he has a man with a pneumoconiosis with pneumonia and he was grossly disabled. Well, I have seen that in London. He was disabled when he came in. I think he had disabling pneumonia, whereas you might call it temporarily disabling pneumoconiosis.

I want to quarrel with his suggestion that the coal macule is not as opaque as the silicotic nodule. The coal macule is smaller than the silicotic nodule. I believe the radiopacity is at great as collagen and the silicotic nodule is larger than the coal nodule, displaces more air and is, therefore, more visible.

And lastly, I want to ask him one very important question, that is in this tubercular negativity of cases of massive fibrosis. That we have never observed. We have found cases of massive fibrosis that only react to one in a thousand tuberculin and I believe we have had one go up to one in a hundred but never a complete negative, and I would like to know what strength of tuberculin he used for

that negative test. Thank you very much, Doctor Sander.

BY DOCTOR SANDER:

Thank you, Doctor Fletcher. Do you want to make your remarks now? Doctor Vorwald has a few remarks to make to correct a false impression of this morning, right?

BY DOCTOR VORWALD:

Yes, I should like to do exactly what Doctor Fletcher did, so that we do not leave a false impression with you.

First, in showing the slides stained for elastic tissue, Doctor Hussey called my attention to the point that I did call it elastic tissue rather than reticulin within the lung, and so I wish to correct that.

The second correction which I should like to make, being a correction, at least attempt to modify the impression which I left with you, and that is with respect to this focal emphysema and I demonstrated a series of cases of workers exposed to dust other than coal dust, where there was focal emphysema, and called attention to the fact that this focal emphysema was then not unique for the coal miners' lung.

In presenting these cases, such as from the granite worker, and from the limestone worker and from the graphite worker, I wish to impress upon you that those were the very isolated cases, that it has certainly not involved

the large group of individuals supposed to be exposed to these dusts.

Furthermore, with respect to the magnatite worker and the hematite worker, the incidence of this focal emphysema, about the foci of dust, is present probably to a greater degree than is present in the graphite worker in the other cases, and to a lesser degree in the coal miners' lung, but that does not mean that with the deposition of graphite in the lung, with the deposition of magnatite, with the deposition of hematite, and indeed with the deposition of limestone, that there must also, therefore, be present a focal emphysema and thus, therefore, the man - the patient must also have a respiratory difficulty. I think that is fallacious and I don't wish to leave you with that impression.

If I may go on and comment just a bit about Doctor Friedman's remarks, certainly Doctor Friedman has had a great deal of experience with the coal miners. I should like again to take a bit of exception with him on the - his statements with regard to the matter of progression, that it is of relatively little concern. He mentioned that it is of little concern whether it be due to massive local concentration of dust or whether it be due to a complicating infection.

I think it is of concern. We must know whether

these coal miners' lungs are more susceptible to infection than is the lung of a worker with deposition of other kinds of dusts.

Again, I should like to make a slight comparison as did Doctor Fletcher, that is, he spoke of emphysema, and that it is important to know the incidence by age also of emphysema, in the entire working population of the coal miners, and to compare that incidence with the incidence in the non-exposed population, by age and by racial characteristics. I think that is also very important before we conclude that emphysema, in the coal miners, about which he has seen, as a common denominator of all of the coal miners exposed and that it is higher than the incidence in the non-exposed population.

With respect to his last remark, that is that he will not only have to know whether these coal miners come from Alabama, and whether they come from Jefferson County and Mine Number 9 and the right side of the truck, I think that's precisely what he has to do, and we all have to do that, and furthermore, I should like to know, and I think we have to extend it, we want to know whether he comes up the track or down the track. We want to know whether that track is outdoors or indoors.

I think those are the things that we must know if we are to solve some of the problems relating not only to

the pulmonary changes in coal miners, but also the pulmonary changes in workers exposed to other dusts.

One more comment and that is with respect to the Roentgenogram and the pathological manifestations of the disease; it is true that the pathology, the pathologies, - the pathologist frequently sees more disease in the lungs than manifested in the chest Roentgenogram. But I don't believe that that should be used as an argument to support the view that there is always something more in the lung than is present in it, than is visualized in the Roentgenogram. Should we follow that reasoning, then we could all - we should say then also, well, this Roentgenogram is negative, it shows no evident change, but there is something more in the lung than what we see in the Roentgenogram. Therefore, this man must have pneumoconiosis, because he had an exposure to coal dust, so we throw the Roentgenogram out the window, we throw the physical examination out of the window, and all we want to know is did or did not this man have an exposure to dust. If he did, then he belongs to the category of pneumoconiosis; if he had no exposure, then we may question. Thank you.

BY DOCTOR SANDER:

We already have a few questions of Doctor Friedman. I'm going to ask one and then I hope we'll have some more from the floor and then we'll give him a chance to

answer them.

I'd like to know how he makes a diagnosis in a man who has been a miner for some twenty-five years, whose chest film at age sixty-five, when he's retiring, shows only emphysema, nothing beyond that. What - how does he go about making the diagnosis beyond the hot tub test and the - the history of his not feeling well when he gets up in the morning? What do you have besides his history, and possibly the lung function test, which undoubtedly will show a low maximal breathing capacity, because he has emphysema? Anyone who has emphysema has a low maximal breathing capacity. At age sixty-five it's lower than it was at age forty-five, and if you have the additional emphysema, it's going to be still lower. How does he make the diagnosis of coal miners' pneumoconiosis with that situation in mind?

Do we have some other questions? Doctor Kline?

BY DOCTOR KLINE:

Doctor Friedman, I didn't come in at the very beginning of your paper and if you've answered my question, I'm wasting your time, but you talked about cardiac failure in these cases, and you mentioned that before one gave digitalis or treated the patient with the other methods that we have available for the cardiac failure, one should do something about the pulmonary condition. I presume you were referring to McMichael's work in England on the effect of

digitalis on the veins of the body. This work has not been widely accepted, and not generally confirmed, but I'm not taking any exception to that. I am asking this question. What does one do about the primary pulmonary condition? The disease of the heart is the result of increase in the pulmonary artery and the right ventricle. Do you have some therapeutic tool which corrects this condition before correcting the cardiac disease?

BY DOCTOR SANDER:

Any other questions?

BY DOCTOR AMBERSON:

What was the time element between the last X-ray film and the last post mortem examination? -- Doctor Amberson -- This calculation effects me as batting about ninety-seven percent, which is pretty good in any league. The time element between the last chest film and the post mortem examination.

BY DOCTOR SANDER:

In which case?

BY DOCTOR AMBERSON:

I think there were two or three.

BY DOCTOR SANDER:

He had two or three. Any other questions?

BY DOCTOR BRISTOL:

I'd like to ask Doctor Friedman if he thinks it's

possible from the X-ray to make a diagnosis of focal emphysema, number one, and X-ray alone, that is; and number two, do you believe that emphysema is a complication of simple coal workers pneumoconiosis?

BY DOCTOR SANDER:

Any other questions of Doctor Friedman?

(No response).

You have the floor, Doctor Friedman.

BY DOCTOR FRIEDMAN:

On my way up again. I should have stayed home. I'm really surprised. I thought it would be worse than that. I don't know whether I'm going to answer these questions in order and I don't even know whether I'm going to answer them, but I'm going to say something about each one.

As regards Doctor Fletcher's remarks about the incidence of spontaneous pneumothorax, that's true, it's clinicians' incidence of the disease and in my particular situation, I am not in a position to compare a comparable control group of coal miners whom I study, because the people who come to see us, sick coal miners, we don't get a cross-section of the coal mining population.

However, I do think, Doctor Fletcher, that the incidence of spontaneous pneumothorax, even without the comparison, appears to be higher than in the normal population group. Of course, I know that Doctor Goffe, and you

come from that school of Doctor Goffe, feels that spontaneous pneumothorax probably is unrelated to the disease because of the location of the blades, and when I made that statement, I knew I was on dangerous ground. I thought you'd overlook it.

Regarding the tuberculin testing, we use one to one thousandth to begin with and we end up with one to one hundredth and if you're ever down in Alabama, I'd like for you to come down and see some of our negative skin tests, because they really complicate the picture when we can't obtain a positive reaction, and we frequently test them again in three months or six months.

And then, about the location, then Doctor Vorwald made a remark about the incidence of emphysema in coal workers. I took one hundred patients from the age forty to sixty in our own clinic, random sample, men, and then I took a group of coal miners, forty to sixty years of age, and compared the incidence of emphysema in both groups.

Now, I admit before I go any further, that it's not a fair comparison because the people in the control group who come between the age group of forty and sixty, came in complaining perhaps of some heart disease or gastric disturbance or something of that nature, but the coal miners all come complaining of something wrong with their lungs, but at least in that group, I would like to give you the

benefit of what we found.

We found less than ten or twelve percent emphysema in a control group whereas in a group of coal miners who came to be studied, and we used maximal breathing capacity, both lung capacity, fluoroscopic examination, X-ray, we didn't use any gas studies, we're not equipped for it - we found that the incidence of emphysema by the most rigid standards in a coal mining standard, in the age group of forty to sixty was in excess of seventy-five percent.

I admit, to begin with, that that's an unfair comparison, but as I say, I don't have a cross-section and I wasn't able to X-ray all of the coal miners in an area whether they were sick or whether they weren't.

I'm glad that Doctor Vorwald felt that the location of the man's work was important. We all realize that, and I think we have to realize, not only the geographical location of the mine, but where he works in the mine, whether he's a timberman, whether he's a short fire man, whether he does drilling or whether he does overhead pinning, these are all important factors, and those of you who may not be familiar with some of the more modern methods of coal mining, I would like to tell you, and Doctor Fletcher made some remark touching on that this morning, in this machine age of mining we're beginning to see patients in the lower age groups who, after lower periods of exposure as coal workers,

are developing pneumoconiosis by Roentgenographic change and functional changes. It's because the machines don't know to cut through just coak, they cut through everything and they want to do that so you can get the most out of a seam of coal, and the overhead pinning, that is, of course, the same thing as drilling in pure rock, and you're going to get a pure silica exposure because the purpose of overhead pinning is to fasten the roof of the coal mine and instead of with beams, you're going to fasten it to the overhead strata.

I had the privilege last week to examine some coal miners in Peru, South America. I'm only sorry that they wouldn't let me have the X-rays to bring up here, because what we see in our coal mines isn't anything like what we see there, what we see in their coal mines isn't anything at all like what we see in our coal mines in Alabama and Doctor Fletcher didn't show anything that looked like any Peruvian coal miner, and if you think we have patients, they have many more, because their patients are Indians who live fifteen or sixteen thousand feet above sea level, and to begin with, they have emphysema and then you have to start to eva luate the disability on the basis of that, so we ought to be pretty glad our problem isn't complicated in that manner.

Now, Doctor Sander asked me, I think I have the

age right, in a man about sixty years old, who has worked so many years in a coal mine, who has no Roentenographic changes, but just has evidence of emphysema by our standards, how could I say this patient had pneumoconiosis. I wished I could say so, Doctor Sander, because I'm convinced in my own mind that many of these people do not show Roentgenographic changes compatible with the results of their occupation. I believe that they develop emphysema or rather we should put it this way, I believe just like another sixty year old man, they may develop emphysema in the usual course of life, but they're more prone to develop emphysema if they've worked in a coal mine.

The mechanical effect over a long period of time of the inhalation of non-soluble dusts, the effect in internal bronchial, with infection and without infection, causes an intermittent occlusion over a long period of time of a balance value of nature, and if you do that long enough and often enough for twenty to thirty years, you're going to have some effect on your alveolae, and you're going to have emphysema as a result of your occupation.

The day will come, I'm certain, that we will be intelligent enough as doctors, and will find the correct diagnostic methods to be able to prove that the emphysema we see in coal workers, even without Roentgenographic changes, may be the result of their occupation, taking into

consideration the fact of the usual aging process in man.

Now, Doctor Kline asked me about treating the patient with congestive heart failure and something about why we didn't use digitalis first , and what corrected the pulmonary function before we treated heart failure. I'm not familiar with the man's name you mentioned, that's only my own observation.

We found that we are able, very quickly, to improve the pulmonary function in an individual with the judicial - or judicious use of proper aerosol therapy and oxygen. We treat them for about twenty - about twelve hours for their lungs first. We give them aerosol therapy, oxygen, and then after we have improved their pulmonary function to some degree, we then go ahead and digitalize rapidly with one of the popular new preparations that are designed for rapid digitalization.

The reason we did that was because we had occasion to treat several patients with pneumoconiosis and heart failure and we treated them with the standard methods and we lost a few patients, so we felt that we would give it a try and treat them from the other end first, treat the pulmonary side first. We did and we found that it's been successful.

Doctor - Doctor Bristol asked me how do we establish the diagnosis - will you restate your question; you

know I had a lot of trouble here keeping up with you fellows.

BY DOCTOR BRISTOL:

Well, I believe you mentioned in your paper, that in demonstrating one of your chest Roentgenograms, you pointed to an area of focal emphysema and I asked you the question, do you think we can see focal emphysema on the Roentgenogram.

BY DOCTOR FRIEDMAN:

Well, now, I get it now. Now, when you see a lot of X-rays in coal miners, it's just like anything else in medicine, you get used to seeing something, you think you know something and after a while, when you've had autopsy specimens to go with Roentgenograms to compare, and then you can go back and compare your Roentgenogram with your pathology, then you can see some changes in another X-ray which at least suggest the same thing you saw previously, the diagnosis of focal emphysema is not a definite diagnosis from the Roentgenogram; it's presumptive on the basis of experience.

BY DOCTOR BRISTOL:

Well, would you enlighten me by telling me what characterizes, in your own mind, focal emphysema in the chest X-ray?

BY DOCTOR FRIEDMAN:

Well, in the chest X-ray of focal emphysema, you

start to get the idea you're looking through something like a mesh work, like a dilated sponge. You get the idea that the areas of translucency, surrounded by areas of increased density have a regular nature, but you can not decide, it's a sort of a linear character of the X-ray, you can not make the diagnosis from the Roentgenogram, and it's a presumptive diagnosis, and after you've seen enough of them, I think you can usually guess correctly.

Now, another Doctor asked me about the - about the time element between the Roentgenograms and the necropsy material, is that correct? Now, the Roentgenograms perhaps were thirty - a month before the patient died or a week, it doesn't make much difference, but just within that limit, I think, with the exception of one patient who died, the one with the spontaneous pneumothorax. He didn't die until about two and a half years after we had that X-ray, but I wanted to show the spontaneous condition.

Now, the radiologist didn't have a great deal of difficulty batting ninety-eight or ninety-nine percent, because most of the X-rays that he looked at were pretty easy to diagnose as pulmonary dust disease of coal workers, together with their occupational history. However, as Doctor Bristol pointed out in his question, the X-ray man was not able to tell me, nor am I able to tell you definitely that this man, this patient was going to have focal emphysema

in the pathologic specimen. It's presumptive on the basis of experience.

BY DOCTOR BRISTOL:

Louis, did you forget my question which was, is emphysema a complication of simple coal miners pneumoconiosis?

BY DOCTOR FRIEDMAN:

Emphysema is a complication and, as I said ...

BY DOCTOR BRISTOL:

Simple now.

BY DOCTOR FRIEDMAN:

Emphysema is a complication of simple pneumoconiosis, focal emphysema, but you see bronchitis, emphysema and chronic bronchitis are so closely associated with pneumoconiosis that you should consider the part of the disease entity. You remember I showed the pathologic specimen of the degeneration in the individual, and you saw that that was a definite change in the pathology and Doctor Vorwald demonstrated that this morning too.

BY DOCTOR SANDER:

We're - thank you very much, Doctor Friedman. We're a little bit late, but I think a two minute stretch would help all of us.

(Recess taken from 3:30 to 3:50 P.M.)

BY DOCTOR SANDER:

Will you take your seats please? We have two very interesting papers remaining this afternoon, the first one by Doctor Hurley Motley, recently of Philadelphia, who has done the most, one of the most outstanding pieces of work in lung functionstudies and therapy of pulmonary dust diseases. Doctor Motley is now heading for a more salubrious title. He is on his way to Southern California as Associate Professor at the University of Southern California Medical School. Doctor Motley.

BY DOCTOR MOTLEY:

(Doctor Motley read a prepared paper which is on file at the Saranac Laboratory).

BY DOCTOR SANDER:

I think you'll agree that's a lot to digest in forty-five minutes. I don't see how you could cover that much that fast. We'll withhold discussion on Doctor Motley's paper until after the next paper, because they're on essentially the same subjects.

Doctor Philip Hugh-Jones comes to us from England. He has been with Doctor Fletcher's pneumoconiosis research in Cardiff. Doctor Hugh-Jones wants me to state, asked me to state that his collaborator, Doctor Gilson, has worked with him and would be one of the speakers had he been able to come here today. Doctor Jones will give us his experience in the function studies of the coal miners in South Wales.

Doctor Jones.

BY DOCTOR HUGH-JONES:

(Referring to an exhibit on table) Just for the benefit of any security people, that isn't part of an atomic bomb.

Mr. Chairman, Ladies and Gentlemen: The lung function studies I want to talk about this afternoon, as the chairman has told you, were done in collaboration with Doctor Gilson, and we were working under Doctor Fletcher in South Wales where this extreme amount of coal workers pneumoconiosis has occurred.

Now, the most important symptom of pneumoconiosis is excessive breathlessness on exertion. But since this symptom is not specific in any way and neither are there any specific clinical findings of the disease, the diagnosis of pneumoconiosis in life, depends on the radiograph, and all too often, the disease is thought of as a radiological abnormality. However, I know you would agree with me that it is the disturbance of lung function and not the radiographic changes which matter to the man, and the main purpose of our work was to find out the functional significance, if any, of the radiograph.

But before I describe that experiment, I would like to be quite clear as to what this 'coal-workers' pneumoconiosis' is that we see in Britain. Radiologically

and pathologically, it's distinct from silicosis, that is the pure classical silicosis, and we know, for example, from the work of Gough, who found it in the coal-trimmers at the docks, that it does occur, just from the exposure to coal dust only, because those men who load the coal in ships have no exposure to rock dust, and they get typical X-ray findings of coal workers pneumoconiosis, and in the mines, it usually occurs in the worker on the coal face.

Now, the disease we see in Wales, radiologically, is no different, as far as we can make out, from the descriptions of the disease given here in the United States, given in other parts of Great Britain, given in Europe and given in Australia, so I want to be quite clear that I'm talking about coal workers pneumoconiosis, and the results I think are applicable to it in Wales certainly, probably in other parts of the world, but we have no reason to suppose that the studies are applicable to silicosis, which is a different pathological condition.

Now, in describing our work, I shall use the International Classification of the X-ray which Doctor Fletcher told you about this morning, and may I have the first slide please.

And, as you remember, from his remarks, that is divided into the simple pneumoconiosis. There is a typical example of the stage tree of the simple disease, and into

the complicated or conglomerate shadows. May I just have the next two slides. The first one there is a typical Stage B, and lastly Stage D, just to remind us what we're talking about, which shows the extreme distortion -- may I have the next slide, please -- the extreme distortion you get of the lung structure.

Here is the slide, with the trachea moved right across and this large basal emphysema which I think, radiologically, everybody would agree is basal emphysema.

Now, for our work. The main intent, as I have said, was to find the functional significance of the different X-ray stages, but we also wanted to know why coal workers were breathless. May I have the next slide. So I can put down that was our first object of our experiment, the cause of the breathlessness and its severity in relation to the radiograph.

Now, the second intention was a more ambitious one, and that was to try and find out the interrelation of the different lung function tests we were using, that is to what extent one test overlapped with another, and what precisely they were measuring in the lungs.

Now, I shall deal this afternoon, with our first object and the second I will put out as a demonstration for the results of that. Now, of course, many other workers have tried to get out this first object, but a review of

the literature has shown us that very few people claimed that there is much relationship between the radiograph and the functional state of the map. But also a review of the literature convinced us that workers who have claimed to have found this lack of relation had very little real power to make that conclusion.

And I have put up here, and I hope you'll forgive me, what I think are the necessary criteria before one can accept any work relating the X-ray to the functional state of a man. First of all, it must be an unbiased sample and a hospital population is particularly unsatisfactory from this point of view, because obviously, the breathless man comes up to the hospital, and I would disagree with Doctor Motley when he says that his patients were unselected. He may have not selected them, but when he draws a conclusion that there is no relation between the X-ray and the functional state of the man, I would submit that his work is done on what is, inevitably, a biased sample, if it were volunteers coming up to a hospital.

Secondly, there must be allowance made for other independent variables which everybody will agree effects that function besides pneumoconiosis. The obvious one is age, but there are many others.

Thirdly, there must be adequate functional tests and we must know their accuracy, their reputability, and

what I have called their discrimination, that is their power of segregating the normal from the abnormal. I will explain that later if you people are interested.

And finally one requires precise radiological description. My own impression of the literature is that physiologists have gone to a great deal more trouble over their physiological tests than they have over the radiographs and there are very few physiologists who are meticulous enough to accept carefully graded tests to give the same criteria of excellence to their radiographic selection.

Well, bearing those points in mind, we designed the following experiment -- may I have the next slide, please -- which was designed to try and meet those requirements. We selected, at ages thirty-five, forty-five and fifty-five years approximately, eight men in each of the different radiological categories, normal working man, that is men with less than simple pneumoconiosis, men showing Category 1 or 2, men showing Category 3 or 4, B and the D's.

The men were selected entirely on their radiograph, on no other basis at all and the radiographs were classified and read in duplicate, by independent observers. Furthermore, we also selected, as another control besides the control miners, we selected forty men who were non-miners and we chose them simply to be sure whether mining

itself might have an effect on lung function as opposed to the radiograph.

Well, now, besides all these men, we did, as you will see in the talk, select some other workers as other controls or some other men. We selected five cases of advanced non-industrial emphysema, chosen for use by Professor Christie in London, who sent us them as undoubtedly cases of emphysema, and we also selected two rather reare cases of a curious fibrosis in the lung, where it was thought that there would certainly be a gas transfer defect. That is those other cases were simply put in as controls to make sure that the appropriate lung function tests did measure what they were meant to. That is, if we got negative findings in pneumoconiosis, we wanted to be sure that the tests themselves were not at fault.

Now, before the men were tested by ourselves, they were examined cardiologically and clinically, and the clinicians were two independent observers, who not only examined the men, all these men, a hundred and sixty-five cases, but they graded them according to their degree of breathlessness.

May I have the next slide, please? And their breathlessness was graded purely symtomatically on a series of questions as to whether they were undoubtedly as good as other men when they were walking about on the flat, whether

they were able to walk with men of their own age on the level, but they got behind on hills or stairs, whether they were able to keep up with menon the level, but they dropped behind after a long time, or whether they could only walk fifty yards or finally whether they were breathless at rest.

Now, such a classification is obviously open to observation rather, but when two independent clinicians did it and we have found that such a grading of breathlessness does at least enable one to define men into, shall we say, four categories of breathlessness with which to compare lung function studies.

Now, for our own tests. May I have the next slide please? I haven't got time to go through in detail all of these tests, but I think and hope they're fairly familiar techniques. The exercise test was a stepping test in which every man did work at the rate of 350 killogrameters a minute, and we measured the amount of air he needed to do that standard amount of work. We also, in some cases, measured the arterial oxygen saturation of the blood before and after the exercise.

Now, the exercise was, in fact, similar to the sort of exercise that one gets on a bicycle or a pedometer, in that it was independent of the weight or size of the man. That was standardized out. We measured, as you see there, maximum voluntary ventilation and we also tried to determine

whether a man had any bellows spasmor not, by using adrenalin.

We measured the arterial capacity, using our closed circuit, which compares very well with the open circuit technique described by Doctor Motley here and first put out by Doctor Kellner, and finally the last test in which we tried to find the intra-pulmonary gas distribution and the rapidness of gas transfer into the blood. I must spend a little time on that.

Now, our men, as I told you, were not volunteers coming up to the hospital. They were men selected at random from the whole of the coal field, many of them working miners, and we would, therefore, think, that it would unjustifiable to do arterial puncture on them, and so - and we wanted to try and get a test which would give us the information with as little as possible in the way of disturbance to the man, and we chose to give them an inhalation of air in which some of the nitrogen was replaced by helium simply to act as a trace gas, and in which there was a small quantity of carbon monoxide introduced.

The idea was that helium goes into the lungs and is distributed without going through into the blood stream. It's the less soluble of all gasses in the blood. The carbon monoxide on the other hand, goes across into the blood stream, and in lung concentrations, exerts negligible back-tensions. May I have the next slide, please?

You will see here the results, the net results of the test. Here I am plotting up here the concentration of gas that the man breathe out after he stops inhaling the mixture here. And you will see that by about forty breaths or fifty breaths, a normal subject is breathing out what he is breathing in in the way of helium. On the other hand, the carbon monoxide comes up and then flattens out. The difference here being due to the rate, due to the absorption of the carbon monoxide into the blood stream.

Now, the results. As you see, there, they are not very helpful and they do need mathematical analysis in the form and components of these curves, and I can't go into that now, but I would like to tell you that in normal subjects, the helium distribution curve in the blood and in the lungs is the sort of thing you would expect if it followed through the lungs. That is an exponential curve, and in emphysema, it seems, it ventilated much more quickly and washed out of gas much more quickly than other parts, so that instead of being a single exponential, it takesthe combination of two exponential curves, and the mathematical analysis is entirely different.

It takes much longer for the man to replace the gas in his lungs and the replacement is in a different form, and we simply express that in terms of two mixing indices which I shall describe and they do represent the

degree to which the lungs are unevenly ventilated. That, I shall use later on, as a physiological definition of emphysema.

The carbon monoxide is expressed as the proportion of gas removed from the inspired gas, because that gets over the difficulty of the different ventilation of different subjects when expressed like that.

Now, for the results. May I have the next slide, please? Here you see the standardized ventilation. That is the amount of air the subjects need for doing a standard amount of exercise, and here are our different radiological groups and you will see that a-apart from a few men lying outside these lines, most of the men did not need a particularly increased amount of air compared to the normal subjects, to do the exercise.

In fact, in other words, they had only about a twenty percent rise, this is about twenty percent here, greater than the normal, in the most abnormal subjects, in their ventilatory requirements for a given amount of work. So it's perfectly clear that the cause of the breathlessness is not primarily an increased need for air when doing a standard amount of work, in pneumoconiosis.

If, on the other hand, you look here at the maximum voluntary ventilation or if you like, the maximum breathing capacity, that represents the maximum output of

the pulmonary bellows. It tells you how much air a man can pass in and out if he tries his hardest and you will see that, first of all, there is a very marked falling off of the maximum voluntary ventilation with disease, but secondly, there is a very wide scatter about this average and if you look at this diagram, these crosses represent the thirty five and the circles elderly men and you will see there is a big age effect. Mostly the young men are at the top here and the old men are at the bottom, so now if we take out this effect of age -- the next slide -- and you see the results now of the mean maximum voluntary ventilation of those different radiological groups against the age in years, you will see that normal men drop their maximum power of their lungs as they get older, certainly men with simple pneumoconiosis, but you will notice that simple pneumoconiosis or the degree of it doesn't make much effect.

The 3's are just as bad as the 2's and even the none, without pneumoconiosis, or shall we say without any radiological pneumoconiosis, are worse than the other men, but not seriously worse, but when you come to the complicated disease, you will see that they have a marked falling off of the maximum ventilatory power of their lungs, particularly in Group 3.

May I have the next slide, please? Here is the result really shown in another way. It's the form of a

solid histogram. You will see the base of it is our original experimental plan. Here are our different age groups and here are our original - here are our different radiological groups on the bottom and the height of the groups represents the average blocks at the top. On the top of the clinician's effect of their breathlessness done entirely independently of the history, and the bottom it says 'dyspneac index used by McCann', in which we express the amount of air a man had to use as the amount of exercise in proportion to the amount he conceivably could use.

Now, it's obvious when you stop to think about it, if you're using all the air your lungs can take in and out, you're feeling extremely breathless whereas if you're only using about twenty percent of it, you're not feeling very breathless, and this index we do find agrees remarkably well with the assessment of breathlessness given by the clinicians, but it's a more sensitive test. It's not surprising, you can measure the thing better than you can - well, I wouldn't like to say guess, I'll say estimate it, and you see here that the normal subjects get slightly more breathless with age, but the important thing is that simple pneumoconiosis, although it puts a slight premium on normal aging, doesn't have much effect until the men are very old, that is, fifty-five years, but the complicated pneumoconiosis puts this marked premium on aging and

furthermore, this Group D, the rate of change with age is very much steeper.

May I have the next slide, please? Now, Doctor Motley expresses most of his results in the terms of residual air percentage of the total lung volume, and here I know you will forgive me, if I disagree with him to some extent. First of all, in some of his papers, and he did this afternoon express his other results in terms of this one test, and it does seem to me that automatically then, that test is labeled as a hundred percent and everything else is worse than that, and I don't quite see why he chooses that test for emphysema and not some others.

Secondly, I would like to show a difference in our results than the ones he got. Here is the residual capacity percent and you will see that in the pneumoconiosis cases, there is a rise, but not a very marked rise. There is a rise, you will note, with age in normal subjects and there is a slight increase with disease particularly, in the group D, but it's nothing like as marked as in the non-industrial emphysema people and the - there is one slight point here, and that is with regard to hospital populations.

When we took a hospital population, we found exactly the same results as he did, that at least fifty percent of the men had a large residual air percentage in that

case, whereas here only about nineteen percent had one in the general population with pneumoconiosis.

May I have the next slide, please? Furthermore, I would like to analyze the meaning of this residual air percentage. Here you will see the absolute figures expressed, and what's noticeable here is not so much the rise of the residual air itself, but the fall in the total lung volume, and I believe you're seeing in pneumoconiosis exactly the same thing by looking at the residual air percentage as you are in measuring the M. V. B. or the fall in the vital capacity.

The percentage in pneumoconiosis is, by and large, raised because of the fall in the complimentary air, whereas in some of the non-industrial emphysema, both the residual capacity is increased, and the total lung volume increases, so that the percentage is still up, but you may get a rise of residual air percentage from two different defects, one from an increase in the absolute volume and you get that, notice, in Category D pneumoconiosis, where I believe they are developing true hypertrophic emphysema on the radiograph, whereas in our people it looks to me as if it was due to a fall in their bellows, namely, their vital capacity, rather than a rise in the residual air.

May I have the next slide, please? Now, we've mentioned this term emphysema. Well, we might discuss

afterwards what we mean by emphysema, but the one thing is clear cut and it's been demonstrated very ably in Philadelphia in the States by Fowler and Comroe and his group there, that in this emphysema, you do get this inequality of mixing of gasses in the lungs. We find exactly the same thing, and this is the result of this helium mixing index which expresses the primary exponential - the binary exponential replacement of gas, and you will see that the normal binders, if I may call them that, are grouped with the normal subjects, truely normal. The simple pneumoconiosis are grouped with Category B, and it's only Category D that you get a very marked falling off of the index.

If you compare that with the advanced non-industrial emphysema sent to us from London, you will see that the test does show a marked effect in non-industrial hypertrophic emphysema. Now, it is tempting to suggest that this might represent what Gough describes pathologically as focal emphysema, because after all, there is no reason to see why a massive shadow per se, in this group B should alter the gas distribution in the lungs, and it is equally tempting to suggest that this corresponds with the large fuller emphysema of the radiograph. Anyway, we have not got enough pathological correlation to make that point clearly. Nevertheless, I think the results are suggestive.

May I have the next slide, please? Now, lastly,

what about the gas transfer defect in pneumoconiosis? Well, do you remember when I was talking about the exercise results, I did say that there was a rise of about twenty per-cent only, in the amount of air for a given amount of work and that would, to me, suggest an indication either of gas distribution in the lungs or gas transfer being deficient. Here are the results of the carbon monoxide uptake, and you will see that there is not a marked deficiency of carbon monoxide uptake until you get down to the Group D, and even then it isn't very great and when you remember the scatter about these averages, you'll find that the results here are barely, of bare significance.

These are the Category D as we have had before; those are the Category B and those the simple pneumoconiosis, but to show you that that test does reflect gas transfer defects when it occurs, here are these cases of rather rare fibrosis which have been published recently in the Medical Journals, and you will find that these men were - with curious fibrosis in the lungs, do take up about twenty percent of the carbon monoxide in the mix-up, whereas these people take up about seventy percent.

So I think you can say that gas transfer is not a marked effect in pneumoconiosis except in its later stages and then it is often associated with heart disease and cardiac failure in our experience, and I think that

result does fit in with the clinical observation that cyanosis is not a common feature of pneumoconiosis. It's breathlessness on exertion these men get, and not breathlessness at rest nor cyanosis.

Now, May I just try and draw some conclusions about this work, and to suggest to you some of its implications. First of all, it does seem to me that we have established on the average, that there is a relationship between the X-ray and the functional state of the man. Notice, I said on the average and in groups, and I think the reason why people have missed that relationship is because they have been working with bias populations and they have not taken the effects of age into account.

I don't know whether you remember that the slide I put on of the dyspneac index, did show that young men with - even with Category D pneumoconiosis, may not be very breathless whereas the old men may be extremely breathless, and if you read an X-ray in conjunction with age, you get an idea on the average, of the man's breathlessness, but that doesn't work in individuals, because the scatter about the average, and you do need functional tests to precisely say how disabled a man is, and no normal amount of reading of the radiographs helps you. On the other hand, the relationship to the radiograph, I think, is important from the point of view of prevention of the disease. We have shown that

it is the common disease which is the disabling disease, whereas the simple pneumoconiosis, whatever it's degree, and notice I say whatever it's degree, does not produce serious disability.

That gives one a guide as to how one can check the efficacy of dust suppression in the mines. It means that one can afford to let a man develop the very earliest stages of simple pneumoconiosis without thinking that he will be seriously disabled by it, and you can then say to the engineers, look, this mine is not safe and if the man goes on, on repeated radiological examination progressing, he will be liable to serious disability, by catching, ir you like, or by developing progressive massive fibrosis and complicated disease, and the epidemiological studies have shown the complicated disease, whether it's due to tuberculosis or not, I don't know, but in itself, does not occur, and this is a significant and marked - at least Category 2 simple pneumoconiosis in the radiograph, so I think the practical importance of this work is in relation to prevention of the disease by periodic X-ray examinations.

Then, lastly, there is the question of compensation and disability, which I know many people will be asking. Well, first of all, there is the very intriguing and slightly worrying result that we found, that the normal miners, with less than Category 1 pneumoconiosis in their

X-ray, were significantly disabled compared with the truely normal non-miners, and that was confirmed in a pit where men were working underground and compared with non-working population, but obviously, that's got to be done on much larger groups of men in other industry, heavy steel industry, farming workers, shall we say, before we can make an assertion, but it is interesting that, shall we say, make a guess if you like, that breathlessness can occur in miners, compared with non-miners, to some extent, and one might argue that the disability is the thing on which the functional tests should give a precise answer.

The loss of faculty, if you like, is what the clinician has to assess, as the meaning of the functional tests and I think, personally, compensation should depend on that.

Lastly, I have said nothing about the interrelation of the tests which was the other object of our work. Now, Doctor Motley, for example, suggested that a battery of three tests, vital capacity, maximum breathing capacity and residual breathing capacity percentage, could form a useful measure of ventilatory function. It - perhaps I understood - or misunderstood Doctor Motley, but it did seem to me that he compared them individually with the sum total of the three. I would like to get his comment about that.

But what we have tried to do, instead of arbitrary selecting tests, is to try and find the precise mathematical relation to our problem and this rather curious looking obeject here which I have put out as a demonstration does show that. The angles between each of these knitting needles represents the degree to which tests are measuring the same thing. It's all right - this will be out on demonstration, you can see it somewhere else.

If tests are at right angles, it means they're entirely independent, measuring something to the - something totally unrelated. The nearer they come together, the more they're measuring the same thing, and we have also tried to relate those to the fundamental properties of the lung, namely, the bellows, action, the size of the lungs, the distribution of gas and gas transfer and we believe, through this method which can be got out, is an interesting one in doing two things: One, in enabling people to formulate a logical battery of physiological tests because you obviously don't need to use two testw which are measuring the same things; secondly, it does show you in relation to the X-ray and age, the type of physiological lesion which onegets in coal workers' pneumoconiosis.

(Applause).

BY DOCTOR SANDER:

Thank you very much, Doctor. I'm going to ask

Doctor George Wright to open the discussion. Will you come up, George, please? Doctor George Wright of the Trudeau Foundation who has done all the lung function work here at Trudeau. You going home?

BY DOCTOR WRIGHT:

Yeah, I was just on my way home.

It was a pleasure for me to say a few words about these last two papers. This is, of course, a field that I am intensely interested in and I suppose some of you had a bit of difficulty in following the enormous amount of data that was presented. I must confess to having had the same difficulty.

This sort of work does not lend itself well to easy digestion, and takes sometime going over in order to come up with an intelligent discussion. I shall confine my remarks, therefore, to some generalities.

First of all, most of the questions that I had posed for Doctor Motley have been posed for me by Doctor Jones. I believe that conditions here are a little bit different than in England. I can only envy Doctor Jones' opportunities to make a study of a group of individuals which embraces all types of experiences, in which he can set up his experimental approach and before he begins to get his men.

In our country, we're pretty much forced to take

the cases as they came, and I think that Doctor Motley would be the first to say that his material is not necessarily representative of the industry as a whole, but so far, we have not been permitted to go into a field and take all of the men or sample them in a random fashion, and I think perhaps the finest thing about Doctor Jones' presentation is that that is precisely what he has done. I wish that we had an opportunity to do that here.

I have some little doubt about this term breathlessness. All of us are seeking for some final criterion of a man's ability to work. Breathlessness is not a simple thing. It is an extremely complicated one. Those who practice medicine know that. The psychological aspects of exertional dyspnea are enormous, and I wonder whether or not breathlessness is an adequate criterion of a man's ability to work. It is certainly one of the things, but it should be subjected to the same scrutiny that Doctor Jones has subjected his X-rays and other tests to.

Now, at the end of this day, now, I had hoped that I would learn whether or not the coal problem in England was the same as that in the United States. Now, I must confess that I don't know whether it's the same. Now, I've had no experience with coal miners' disabilities, but it would appear to me from the paper of Doctor Motley and also Doctor Jones, that the disabilities are essentially the same

as that that has already been found and well described by McCann's group, by ourselves, and by others abroad, in silicosis that occurs in metal miners or foundry workers, in that the discreet nodulation and the linear exaggeration type of abnormality, little in the way of disability is disclosed, and that it's only in the conglomerate disease that one meets moderate to severe grades of disability. If I'm wrong in this, I would welcome correction.

I had wondered whether or not the changes in the coal mining industry were distinctly different from that in other industries. I still don't know, however, from the papers given by our American coal workers, as to whether or not what they are seeing in the coal industry is the same thing that is being seen by Doctor Fletcher's group in Britain.

I want to make one thing very clear. I know full well the enormous amount of work that has gone into these two papers, and I think the two men are to be congratulated on the very fine piece of work. I'm sure that as the years go by, we - they will be deriving more and more good information from the data that they already possess.

(Applause).

BY DOCTOR SANDER:

Do I hear any other comments from the floor or any questions? I'm sure you all have questions but you probably

don't want to ask them. No questions?

(No response).

Doctor Vorwald has a few announcements to make. Thank you very much for the excellent papers.

BY DOCTOR VORWALD:

Members of the Symposium, during the course of the stay, you've heard some outstanding studies. I merely want to say many is the man who has pointed to the fact that we need more studies. I plead with you to make it available, make these studies available to us here, elsewhere in this country, whereby we might come further, come closer to the solution of the problems confronting us with respect to coal miners, coal workers and the pulmonary changes which they show. I think You will join with me in complimenting the speakers of today - Doctor Motley, Doctor Fletcher, Doctor Jones, Doctor Hugh-Jones, for the contribution which they have made to this Symposium. Thank you.

BY DOCTOR SANDER:

I'm very sorry I didn't give Doctor Motley and Doctor Jones an opportunity to answer the questions which were asked. Will you take over for a minute, Doctor Motley?

BY DOCTOR MOTLEY:

With regard to Doctor Jones' question on emphysema being a reduction in vital capacity only, that is not our experience. We find that the vital capacity is reduced,

but we find also that the absolute residual volume is increased. In our fourth group, the ones for advanced impairment, it's more than a hundred percent increased, that is the predicted residual volume is thirteen hundred; it's over twenty-six. In some instances, we have residual volumes as high as forty-five hundred c.c.'s. True, the vital capacity may be only two thousand, but we have the absolute increase in residual volume.

I did perhaps not make it clear in my evaluation of emphysema, we have really three things, the residual nitrogen percent in the expired air at the end, after breathing oxygen for seven minutes - if that is elevated, that is significant; secondly, the absolute volume as a residual volume with respect to the normal predicted, and thirdly, the residual predicted on volume.

I hoped - I tried to make it clear, I mean to point out the correlation with that arbitrary classification. When the residual volume is more than thirty-five percent of total on volume, lung volume, we have a fairly nice correlation with direct blood studies, getting rid of the carbon dioxide and lower, of the air in the air sacs as a result of the increased volume of residual air.

The ventilation factor, Doctor Jones, is simply a help before I analyze all my cases with respect to this figure. I used to evaluate independently, the residual,

the maximum breathing capacity. This simply gives me a figure. Now, by and large, if that is from eighty to a hundred, that includes a slight group,from eighty to sixty are moderate, sixty to forty an advanced and below forty far advanced.

Now, the only thing that upsets that, if the oxygen saturation rocks, and I think it is most important that we have blood saturations, because as I pointed out, we have normal resting saturations that may go down ten percent or more with exercise. That indicates disability. If that man is trying to work, he is working with a reduced oxygen saturation, a temporary degree of impotency, which we know increases arterial blood pressure, increases pulmonary vascular resistance.

Doctor Wright's question regards a conglomeration for total disability. In general, we find that is true. However, we do have some cases that would fall in Stage 1 or 2 or even in some, you would call it, normal X-ray, with a history of thirty-five or forty or more inside exposure, are totally disabled. We have found just as severe emphysema, Doctor Jones, in our coal miners as we have found in idiopathic emphysema in non-coal miner groups; some of them have gone up as high as seventy-five percent.

Doctor Jones, I would like to ask you why you use air consumption, as I understand that, that is the total

volume of air rather than the actual oxygen uptake on the basis of body surface area. Isn't there a possibility of air in there due to individual ventilating alveolae that are not profused insofar as arterial punctures go? We don't have any more hesitation in doing them than we do a venal puncture; it's just about as easy and we do that routinely. I have no trouble with our coal miner group.

It is true that we're not able to go out and take the whole coal mine and work it, that is these men have made application. Quite a number of the men that I have studied are still working. They made application to come in for study during their vacation period. We probably have, oh, around a hundred in such a category.

BY DOCTOR SANDER:

Doctor Hugh-Jones, do you have anything to close the discussion?

BY DOCTOR HUGH-JONES:

I probably have said enough, but there are just a few points that do occur to me. First of all, I don't want to sort of take up purely technical physiological points, but I think, Doctor Motley, I might - I must disagree with you again on one point, that you say that if a man's oxygen uptake and his carbon dioxide output are normal, then he's normal as far as his lungs are concerned. I don't think I quite agree with you there.

BY DOCTOR MOTLEY:

Just one part.

BY DOCTOR HUGH-JONES:

It seems to me it rather hinges on Doctor Wright's point about breathlessness and it also raises the last question Doctor Motley asked, why I measure air consumption. Breathlessness itself is a symptom and we were at pains to get somewhat - some way of consultatively measuring breathlessness and it is, after all, we think, the amount of bellows or bellowing and air shifting, if you like, that a man has to do, compared with the amount that he conceivably can do, which gives some measurement of breathlessness or what his sensation of breathlessness is.

I agree with Doctor Motley from the physiological point of view, we are all often interested, for other reasons, in knowing oxygen consumption, and also knowing carbon monoxide uptake which we often remember. The reason for carbon monoxide, there is no way known yet of getting over the back tension in the different parts of the lungs, in evaluating oxygen transfer, but there is the cardinal point which comes out from all this, and that is what is the cause of the breathlessness of pneumoconiosis, and I hope I made it clear that as far as we were concerned, we think it is exertional dyspnea, due mainly to the bellows not being able to have the reserve that they normally should have,

the maximum ventilatory capacity is reduced.

In a few cases, the air requirement, or if you like, the oxygen requirement, but the two don't run quite hand in hand, in increased, but that is subservient to the main cause, which is this diminution of bellowing pwoer in the lungs.

Now, the cause of that, and I think Doctor Vorwald this morning posed what were the causes of breathlessness in pneumoconiosis, and he suggested four, I think, and one was focal emphysema, another one was blocking of the arteries, another one was a ventilation profusion upset, and quite honestly, I think I would like to say that in the main, it's due to a fifth one, which he didn't measure or mention, and that is, I believe myself, that it is mainly a mechanical upset in the lungs, but we don't yet know the answer to this.

The causes of diminution of maximum voluntary ventilation is a thing which badly needs studying. Otis, in America, started a very useful method of thinking about it, and he points out that you can get diminution in the maximum bellows due either to the power of the muscles working the bellows, due to the diminution of the air flow, forms of bronchial spasm, due to a lowering of the vital capacity, which is the stroke of the bellows, or finally, due to the elastic properties of the lungs, the degree of

drag that you get through the actual shifting in and out of the lung substance, and we have not yet got down to that form of analysis, but it does seem fairly clear that in pneumoconiosis, the main effect is an actual reduced size of bellows which one gets and sees in the drop of the vital capacity, and it is interesting in a way, how well vital capacity comes out as a functional test in pneumoconiosis for that reason, I think rather surprisingly well, because it isn't really a dynamic test of function, as you will see from that model.

Then, lastly, there is this next question of what is the role of emphysema. Our results are, and if you will accept them, that's only if you will accept, that bad gas distribution could be defined as oneof the effects of emphysema. Our results do suggest that the focal emphysema described by Gough, is not itself, of necessity, disabling, insofar as we have got, but that the same process, physiological process which gives rise to it, also upsets the elasticity of the lung substance, which diminishes the maximum breathing capacity, but that is speculation.

The whole business of what is emphysema, is an extremely difficult project, or idea, and we are trying to get down to analyzing how one could define physiological emphysema and how that overlaps with what the clinicians talk about and what the radiologists talk about, because

it does seem to me that the word 'emphysema' is shamefully misused in the sense that it is probably a syndrome which may give rise or may arise from various causes within the lungs.

And lastly, there is one point and that is about disability, which I hope I made clear. I did say that I thought conglomerate pneumoconiosis or complicated pneumoconiosis is the main and essentially really disabling part of the disease, but I think you will notice that in the simple disease, as age increased, and by the time people have got Category 3 simple pneumoconiosis at age fifty-five, they were beginning to get fairly disabled.

What happens after fifty-five we don't know. We didn't go above that, and I would stress that our results are limited over those years, because we didn't go beyond age fifty-five, which was the range we were interested in ourselves.

Thank you.

BY DOCTOR SANDER:

Did this discussion formulate any questions?

(No response).

If not, I want to thank all the speakers this afternoon, Doctor Friedman, Doctor Motley and Doctor Hugh-Jones, for this excellent discussion. Thank you very much.

(Session adjourned at 5:45 P. M.)